Refine Search

Search Results -

Terms	Documents
L2 and (suppress$ or inhibit$ or repress$)	56

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L3

Refine Search

Recall Text | Clear | Interrupt

Search History

DATE: Friday, November 24, 2006 **Purge Queries** Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L3	L2 and (suppress$ or inhibit$ or repress$)	56	L3
L2	plzf and (androgen or estrogen) and receptor	56	L2
L1	plzf same (androgen or estrogen)	9	L1

END OF SEARCH HISTORY

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- Ability to resize images for easier incorporation into DialogLink Reports
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- Ability to set up Dialog Alerts by Chemical Structures and the addition of Index Chemicus as a structure searchable database
- Support for connections to STN Germany and STN Japan services

Show Preferences for details

```
? Help Off Line
* * *
Connecting to jane.zara - Dialog - 243084
Connected to Dialog via SMS00205

?  b biotech biochem medicine
>>>W:            76 is unauthorized
         138 is unauthorized
2 of the specified files are not available
```

[File 5] **Biosis Previews(R)** 1969-2006/Nov W3

[File 6] **NTIS** 1964-2006/Nov W2

[File 8] **Ei Compendex(R)** 1884-2006/Nov W1

**File 8: The file has been reprocessed and accession numbers have changed. See HELP NEWS988 for details.*

[File 24] **CSA Life Sciences Abstracts** 1966-2006/Oct

[File 34] **SciSearch(R) Cited Ref Sci** 1990-2006/Nov W3

[File 45] **EMCare** 2006/Nov W3

[File 65] **Inside Conferences** 1993-2006/Nov 24

[File 71] **ELSEVIER BIOBASE** 1994-2006/Nov W3

[File 73] **EMBASE** 1974-2006/Nov 24

[File 94] **JICST-EPlus** 1985-2006/Aug W1

[File 98] **General Sci Abs** 1984-2006/Oct

[File 99] **Wilson Appl. Sci & Tech Abs** 1983-2006/Sep

[File 135] **NewsRx Weekly Reports** 1995-2006/Nov W3

[File 136] **BioEngineering Abstracts** 1966-2006/Oct

[File 143] **Biol. & Agric. Index** 1983-2006/Sep

[File 144] **Pascal** 1973-2006/Oct W5

[File 155] **MEDLINE(R)** 1950-2006/Nov 21

**File 155: The file has resumed updating with UD20061120, with RT=IN DATA REVIEW and RT=IN PROCESS records.*

[File 172] **EMBASE Alert** 2006/Nov 24

[File 266] **FEDRIP** 2006/Aug

[File 315] **ChemEng & Biotec Abs** 1970-2006/Oct

[File 357] **Derwent Biotech Res.** _1982-2006/Nov W4

[File 358] **Current BioTech Abs** 1983-2006/Jan

[File 369] **New Scientist** 1994-2006/Sep W2

[File 370] **Science** 1996-1999/Jul W3

**File 370: This file is closed (no updates). Use File 47 for more current information.*

[File 399] **CA SEARCH(R)** 1967-2006/UD=14522

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[File 434] **SciSearch(R) Cited Ref Sci** 1974-1989/Dec

[File 40] **Enviroline(R)** 1975-2006/Oct

[File 41] **Pollution Abstracts** 1966-2006/Oct

[File 50] **CAB Abstracts** 1972-2006/Oct

[File 103] **Energy SciTec** 1974-2006/Sep B1

**File 103: For access restrictions see Help Restrict.*

[File 156] **ToxFile** 1965-2006/Nov W1

File 156: NLM will not provide updates to the file from November 16-20. Please see HELP NEWS154 for details.

[File 162] **Global Health** 1983-2006/Oct

[File 305] **Analytical Abstracts** 1980-2006/Nov W2

File 305: Alert feature enhanced for multiple files, duplicate removal, customized scheduling. See HELP ALERT.

[File 393] **Beilstein Abstracts** 2006/Q3

[File 35] **Dissertation Abs Online** 1861-2006/Nov

[File 91] **MANTIS(TM)** 1880-2006/Jan

[File 149] **TGG Health&Wellness DB(SM)** 1976-2006/Nov W1

[File 159] **Cancerlit** 1975-2002/Oct

File 159: Cancerlit is no longer updating. Please see HELP NEWS159.

[File 164] **Allied & Complementary Medicine** 1984-2006/Nov

[File 444] **New England Journal of Med.** 1985-2006/Nov W2

[File 467] **ExtraMED(tm)** 2000/Dec


```
?  s plzf (s) (estrogen or androgen) (s) receptor?
Processing
         1978   PLZF
       613416   ESTROGEN
       253700   ANDROGEN
      6112506   RECEPTOR?
S1         55   S PLZF (S) (ESTROGEN OR ANDROGEN) (S) RECEPTOR?

?  rd
>>>W:  Duplicate detection is not supported for File 393.
Records from unsupported files will be retained in the RD set.
S2         22   RD  (UNIQUE ITEMS)

?  show files
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[File 5] **Biosis Previews(R)** 1969-2006/Nov W3

[File 6] **NTIS** 1964-2006/Nov W2

[File 8] **Ei Compendex(R)** 1884-2006/Nov W1

**File 8: The file has been reprocessed and accession numbers have changed. See HELP NEWS988 for details.*

[File 24] **CSA Life Sciences Abstracts** 1966-2006/Oct

[File 34] **SciSearch(R) Cited Ref Sci** 1990-2006/Nov W3

[File 45] **EMCare** 2006/Nov W3

[File 65] **Inside Conferences** 1993-2006/Nov 24

[File 71] **ELSEVIER BIOBASE** 1994-2006/Nov W3

[File 73] **EMBASE** 1974-2006/Nov 24

[File 94] **JICST-EPlus** 1985-2006/Aug W1

[File 98] **General Sci Abs** 1984-2006/Oct

[File 99] **Wilson Appl. Sci & Tech Abs** 1983-2006/Sep

[File 135] **NewsRx Weekly Reports** 1995-2006/Nov W3

[File 136] **BioEngineering Abstracts** 1966-2006/Oct

[File 143] **Biol. & Agric. Index** 1983-2006/Sep

[File 144] **Pascal** 1973-2006/Oct W5

[File 155] **MEDLINE(R)** 1950-2006/Nov 21

**File 155: The file has resumed updating with UD20061120, with RT=IN DATA REVIEW and RT=IN PROCESS records.*

[File 172] **EMBASE Alert** 2006/Nov 24

[File 266] **FEDRIP** 2006/Aug

[File 315] **ChemEng & Biotec Abs** 1970-2006/Oct

[File 357] **Derwent Biotech Res.** _1982-2006/Nov W4

[File 358] **Current BioTech Abs** 1983-2006/Jan

[File 369] **New Scientist** 1994-2006/Sep W2

[File 370] **Science** 1996-1999/Jul W3

**File 370: This file is closed (no updates). Use File 47 for more current information.*

[File 399] **CA SEARCH(R)** 1967-2006/UD=14522

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[File 434] **SciSearch(R) Cited Ref Sci** 1974-1989/Dec

[File 40] **Enviroline(R)** 1975-2006/Oct

[File 41] **Pollution Abstracts** 1966-2006/Oct

[File 50] **CAB Abstracts** 1972-2006/Oct

[File 103] **Energy SciTec** 1974-2006/Sep B1

**File 103: For access restrictions see Help Restrict.*

[File 156] **ToxFile** 1965-2006/Nov W1

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**File 156: NLM will not provide updates to the file from November 16-20. Please see HELP NEWS154 for details.*

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(c) 2006 Royal Soc Chemistry. All rights reserved.
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[File 35] **Dissertation Abs Online** 1861-2006/Nov
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[File 91] **MANTIS(TM)** 1880-2006/Jan
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[File 149] **TGG Health&Wellness DB(SM)** 1976-2006/Nov W1
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[File 444] **New England Journal of Med.** 1985-2006/Nov W2
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```
 ; d s
Set     Items   Description
S1         55   S PLZF (S) (ESTROGEN OR ANDROGEN) (S) RECEPTOR?
S2         22   RD  (unique items)
 ; t /3,k/all
>>>W:  KWIC option is not available in file(s): 399
```

2/3,K/1 (Item 1 from file: 5) **Links**
Fulltext available through: USPTO Full Text Retrieval Options SCIENCEDIRECT
Biosis Previews(R)
(c) 2006 The Thomson Corporation. All rights reserved.
0016018316 **Biosis No.:** 200600363711
PLZF regulates pbxl transcription and Pbx1-HoxC8 complex leads to androgen-independent prostate cancer

proliferation

Author: Kikugawa Tadahiko; Kinugasa Yumi; Shiraishi Ken; Nanba Daisuke; Nakashiro Koh-ichi; Tanji Nozomu; Yokoyama Masayoshi; Higashiyama Shigeki (Reprint)
Author Address: Ehime Univ, Sch Med, Dept Biochem and Mol Genet, Toon, Ehime 7910295, Japan **Japan
Author E-mail Address: shigeki@m.ehime-u.ac.jp
Journal: Prostate 66 (10): p 1092-1099 JUL 1 2006 2006
ISSN: 0270-4137
Document Type: Article
Record Type: Abstract
Language: English

Abstract: BACKGROUND. Promyelocytic leukemia zinc finger (**PLZF**) protein, a transcriptional repressor and negative regulator of the cell cycle, has been characterized as a prostatic **androgen**-responsive gene. DU145 cells show **androgen**-independent growth and lack **PLZF** gene expression.METHODS. We analyzed **PLZF**-regulating genes by DNA microarray using DU145 cells infected with LacZ- or **PLZF**-carrying adenoviruses.RESULTS. DNA microarray revealed that Pbx1 is a prominent suppressed gene in **PLZF**-overexpressing DU145 cells. **Androgen receptor** (AR)-expressing DU145 cells recovered **androgen** -dependent **PLZF** expression and subsequent repression of Pbx1 expression. Immunoprecipitation of Pbx1 in DU145 cells revealed a... ...growth. Double knockdown of both Pbx1 and HoxC8 suppressed cell growth much more significantly.CONCLUSIONS. **Androgen**-independent cell line DU145 cells lack **PLZF** gene expression, resulting in the upregulation of Pbx1 and HoxC8 expression. The Pbx1-HoxC8 heterocomplex may lead to **androgen**-independent growth in prostate cancer.

2/3,K/2 (Item 2 from file: 5) **Links**
Fulltext available through: American Society of Hematology USPTO Full Text Retrieval Options SCIENCEDIRECT
Biosis Previews(R)

0015837623 **Biosis No.:** 200600183018
Genetic pathways in therapy-related leukemia: FHL2 cooperates with del(5q).

Author: Qian Zhijian (Reprint); Mao Liqun; Lebeau Michelle
Author Address: Univ Chicago, Dept Med, Hematol Oncol Sect, Chicago, IL 60637 USA**USA
Journal: Blood 106 (11, Part 1): p 192A NOV 16 2005 2005
Conference/Meeting: 47th Annual Meeting of the American-Society-of-Hematology Atlanta, GA, USA December 10 -13, 2005; 20051210
Sponsor: Amer Soc Hematol
ISSN: 0006-4971
Document Type: Meeting; Meeting Abstract
Record Type: Abstract
Language: English

Abstract: ...and apoptosis in a tissue-specific fashion. Interacting partners of FHL2 include WT1, b-catenin, **PLZF**, and the **androgen receptor**. In subsequent studies, we determined that hematopoietic cells express a novel isoform of FHL2 (termed...

2/3,K/3 (Item 3 from file: 5) Links
Fulltext available through: Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT
Biosis Previews(R)

0015394690 **Biosis No.:** 200510089190
Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF (vol 12, pg 452, 2005)

Author: Buluwela L (Reprint); Pike J; Mazhar D; Kamalati T; Hart S M; Al-Jehani R; Yahaya H; Patel N; Sarwarl N; Heathcote D A; Schwickerath O; Phoenix F; Hill R; Aboagye E; Shousha S; Waxman J; Lemoine N R; Zelent A; Coombes R C; Ali S
Author Address: Univ London Imperial Coll Sci Technol and Med, Dept Histopathol, London, UK **UK
Journal: Gene Therapy 12 (10): p 862 MAY 05 2005
ISSN: 0969-7128
Document Type: Article; Errata
Record Type: Citation
Language: English
Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF (vol 12, pg 452, 2005)

2/3,K/4 (Item 4 from file: 5) **Links**

Fulltext available through: Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT

Biosis Previews(R)

0015283652 **Biosis No.:** 200500190717

Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF

Author: Buluwela L (Reprint); Pike J; Mazhar D; Kamalati T; Hart S M; Al-Jehani R; Yahaya H; Patel N; Sarwarl N; Heathcote D A; Schwickerath O; Phoenix F; Hill R; Aboagye E; Shousha S; Waxman J; Lemoine N R; Zelent A; Coombes R C; Ali S

Author Address: Dept Canc Med, Univ London Imperial Coll Sci Technol and Med, Du Cane Rd, London, W12 0NN, UK**UK

Journal: Gene Therapy 12 (5): p 452-460 March 2005 2005

Medium: print

ISSN: 0969-7128 _(ISSN print)

Document Type: Article

Record Type: Abstract

Language: English

Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF

Abstract: Estrogen receptor alpha (ERalpha) is a ligand-inducible transcription factor that acts to regulate gene expression by binding to palindromic DNA sequence, known as the estrogen response element, in promoters of estrogen-regulated genes. In breast cancer ERalpha plays a central role, where estrogen-regulated gene expression leads to tumor initiation, growth and survival. As an approach to silencing estrogen-regulated genes, we have studied the activities of a fusion protein between ERalpha and the promyelocytic leukemia zinc-finger (PLZF) protein, a transcriptional repressor that acts through chromatin remodeling. To do this, we have developed lines from the estrogen-responsive MCF-7 breast cancer cell line in which the expression of the fusion protein PLZF-ERalpha is conditionally regulated by tetracycline and shows that these feature long-term silencing of the expression of several well-characterized estrogen-regulated genes, namely pS2, cathepsin-D and the progesterone receptor. However, the estrogen-regulated growth of these cells is not inhibited unless PLZF-ERalpha expression is induced, an observation that we have confirmed both in vitro and in vivo. Taken together, these results show that PLZF-ERalpha is a potent repressor of estrogen-regulated gene expression and could be useful in distinguishing estrogen-regulated genes required for the growth of breast cancer cells.

2/3,K/5 (Item 5 from file: 5) **Links**

Fulltext available through: Ebsco Host EJS (Electronic Journals Service) Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT

Biosis Previews(R)

0015126384 **Biosis No.:** 200500033449

Silencing of androgen-regulated genes using a fusion of AR with the PLZF transcriptional repressor

Author: Pike Joanna; Holmes David; Kamalati Tahereh; Davies Derek; Tolhurst Robert; Mazhar Danish; Fishpool Sam; Al-Jehani Rajai; Waxman Jonathan; Zelent Arthur; Lemoine Nicholas R; Ali Simak (Reprint); Buluwela Laki

Author Address: Dept Canc Med, Univ London Imperial Coll Sci Technol and Med, Du Cane Rd, London, W12 0NN, UK**UK

Author E-mail Address: simak.ali@imperial.ac.uk; l.buluwela@imperial.ac.uk

Journal: Oncogene 23 (45): p 7561-7570 September 30, 2004 2004

Medium: print

ISSN: 0950-9232 _(ISSN print)

Document Type: Article

Record Type: Abstract

Language: English

Abstract: The **androgen receptor** (AR) is a member of the nuclear **receptor** superfamily of ligand-activated transcription factors and plays a key role in the development and... ...regulated genes, based on the properties of the transcriptional repressor promyelocytic leukamia zinc-finger protein (**PLZF**). In order to do this, we have made a fusion protein between **PLZF** and AR, named **PLZF**-AR, and show that **PLZF**-AR is able to bring about silencing of genomically encoded AR-regulated genes and inhibit the **androgen**-regulated growth of LNCaP prostate cancer cells. Together, our results show that this strategy is...

2/3,K/6 (Item 6 from file: 5) **Links**
Fulltext available through: USPTO Full Text Retrieval Options SCIENCEDIRECT
Biosis Previews(R)

0015080090 **Biosis No.:** 200400461319
Identification and characterization of PLZF as a prostatic androgen-responsive gene

Author: Jiang Feng; Wang Zhou (Reprint)
Author Address: Feinberg Sch MedDept Urol, Northwestern Univ, Tarry 11-715, Chicago, IL, 60611, USA**USA
Author E-mail Address: wangz@northwestern.edu
Journal: Prostate 59 (4): p 426-435 June 1, 2004 2004
Medium: print
ISSN: 0270-4137 _(ISSN print)
Document Type: Article
Record Type: Abstract
Language: English

Abstract: BACKGROUND. Promyelocytic leukemia zinc finger protein (**PLZF**) was initially identified by virtue of its fusion with RARalpha as a result of a ... ...17) chromosomal translocation that occurs in a small subset of acute promyelocytic leukemia (APL) patients. **PLZF** has been reported to have pro-apoptotic and anti-proliferative activity both in vivo and in vitro. METHODS. Using a modified subtractive hybridization, we identified **PLZF** as an **androgen**-responsive gene in the rat ventral prostate. Northern blot and Western blot were used to characterize the regulation of **PLZF** by androgens in LNCaP cells. Stable transfections of **PLZF** in LNCaP cells were performed to assay the effect of **PLZF** overexpression on LNCaP cell proliferation. RESULTS. **PLZF** mRNA was transiently up-regulated by androgens in the regressed ventral prostate of castrated adult rat. **PLZF** was also up-regulated by androgens, at both mRNA and protein levels, in the **androgen** -responsive human prostate cancer cell line LNCaP. **Androgen** induction of **PLZF** mRNA was not inhibited by protein synthesis inhibitor cycloheximide but inhibited by **androgen receptor** antagonist bicalutamide, indicating that **PLZF** is a direct **androgen**-responsive gene. To study the functions of **PLZF** in **androgen** action, LNCaP sublines stably overexpressing **PLZF** were generated. **PLZF** overexpression inhibited LNCaP proliferation either in the presence or absence of **androgen**, which is consistent with the reported anti-proliferative activity of **PLZF**. CONCLUSIONS. The above observations indicate that **PLZF** is an **androgen** -responsive gene with anti-proliferative activity in prostate cancer cells. Copyright 2003 Wiley-Liss, Inc.

2/3,K/7 (Item 7 from file: 5) **Links**
Fulltext available through: American Society of Hematology USPTO Full Text Retrieval Options SCIENCEDIRECT
Biosis Previews(R)

0013351691 **Biosis No.:** 200100523530

Estrogen-dependent E2a/Pbx1 myeloid cell lines exhibit conditional differentiation that can be arrested by other leukemic oncoproteins

Author: Sykes David B (Reprint); Kamps Mark P
Author Address: Department of Molecular Pathology, University of California San Diego School of Medicine, 9500 Gilman Dr, La Jolla, CA, 92093-0612, USA**USA
Journal: Blood 98 (8): p 2308-2318 October 15, 2001 2001
Medium: print
ISSN: 0006-4971
Document Type: Article
Record Type: Abstract
Language: English

Abstract: ...The cell lines were established by conditional immortalization of primary murine marrow progenitors with an **estrogen**-regulated E2a/Pbx1-**estrogen receptor** fusion protein. Clones were identified that proliferated as immortalized blasts in the presence of **estrogen**, and that exhibited granulocytic, monocytic, or bipotential (granulocytic and monocytic) differentiation on **estrogen** withdrawal. Differentiation was normal and terminal as evidenced by morphology, cell surface markers, gene expression... ...differentiation of the cells could be arrested by heterologous oncoproteins including AML1/ETO, PML/RARalpha, **PLZF**/RARalpha, Nup98/HoxA9, and other Hox proteins. Furthermore, the study examined the effects of cooperating...

2/3,K/8 (Item 1 from file: 34) **Links**

Fulltext available through: American Society for Microbiology USPTO Full Text Retrieval Options SCIENCEDIRECT

SciSearch(R) Cited Ref Sci

13000927 **Genuine Article#:** 839DZ **No. References:** 68

Cross talk between retinoic acid signaling and transcription factor GATA-2

Author: Tsuzuki S; Kitajima K; Nakano T; Glasow A; Zelent A; Enver T (REPRINT)

Corporate Source: John Radcliffe Hosp,MRC, MHU, WIMM,Oxford OX3 9DU//England/ (REPRINT); John Radcliffe Hosp,MRC, MHU, WIMM,Oxford OX3 9DU//England/; Inst Canc Res,Sect Gene Funct & Regulat,London SW3 6JB//England/; Inst Canc Res,Leukaemia Res Fund Ctr,London SW3 6JB//England/; Aichi Canc Ctr,Res Inst, Div Mol Med,Nagoya/Aichi 4648681/Japan/; Osaka Univ,Microbial Dis Res Inst, Dept Mol Cell Biol,Suita/Osaka 5650871/Japan/ (tenver@gwmail.jr2.ox.ac.uk)

Journal: MOLECULAR AND CELLULAR BIOLOGY , 2004 , V 24 , N15 (AUG) , P 6824-6836

ISSN: 0270-7306 **Publication date:** 20040800

Publisher: AMER SOC MICROBIOLOGY , 1752 N ST NW, WASHINGTON, DC 20036-2904 USA

Language: English **Document Type:** ARTICLE (ABSTRACT AVAILABLE)

Identifiers-- ...ACUTE PROMYELOCYTIC LEUKEMIA; **RECEPTOR**-ALPHA ONCOPROTEIN; EMBRYONIC STEM-CELLS; **PLZF**-RAR-ALPHA; SELF-RENEWAL; DNA-BINDING; HEMATOPOIETIC-CELLS; **ESTROGEN-RECEPTOR**; GENE-EXPRESSION; ZINC-FINGER

2/3,K/9 (Item 2 from file: 34) **Links**

Fulltext available through: American Society for Microbiology USPTO Full Text Retrieval Options SCIENCEDIRECT

SciSearch(R) Cited Ref Sci

12798163 **Genuine Article#:** 821DT **No. References:** 35

Reduced intranuclear mobility of APL fusion proteins accompanies their mislocalization and results in sequestration and decreased mobility of retinoid X receptor alpha

Author: Dong S; Stenoien DL; Qiu JH; Mancini MA; Tweardy DJ (REPRINT)

Corporate Source: Baylor Coll Med,Dept Med, Infect Dis Sect,1 Baylor Pl,BCM 286,Room N1319/Houston//TX/77030 (REPRINT); Baylor Coll Med,Dept Med, Infect Dis Sect,Houston//TX/77030; Baylor Coll Med,Dept Mol & Cellular Biol,Houston//TX/77030; Shanghai Rui Jin Hosp,Shanghai Inst Hematol,Shanghai 200025//Peoples R China/

Journal: MOLECULAR AND CELLULAR BIOLOGY , 2004 , V 24 , N10 (MAY) , P 4465-4475

ISSN: 0270-7306 **Publication date:** 20040500

Publisher: AMER SOC MICROBIOLOGY , 1752 N ST NW, WASHINGTON, DC 20036-2904 USA

Language: English **Document Type:** ARTICLE (ABSTRACT AVAILABLE)

Identifiers-- ...ACUTE PROMYELOCYTIC LEUKEMIA; STAT3 SIGNALING PATHWAYS; PML-RAR-ALPHA; ACID **RECEPTOR**; LIVING CELLS; **ANDROGEN RECEPTOR**; TRANSLOCATION; **PLZF**; GENE; TRANSCRIPTION

2/3,K/10 (Item 1 from file: 71) **Links**

Fulltext available through: Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT

ELSEVIER BIOBASE

02986153 2005144680

Erratum: Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF (Gene Therapy) (2005) vol. 12 (452-460) 10.1038/sj.gt.3302421)

Buluwela L.; Pike J.; Mazhar D.; Kamalati T.; Hart S.M.; Al-Jehani R.; Yahaya H.; Patel N.; Sarwarl N.; Heathcote D.A.; Schwickerath O.; Phoenix F.; Hill R.; Aboagye E.; Shousha S.; Waxman J.; Lemoine N.R.; Zelent A.; Coombes R.C.; Ali S.

, United Kingdom

Journal : Gene Therapy , 12/10 (862) , 2005 , United Kingdom

CODEN: GETHE

ISSN: 0969-7128

Document Type: Erratum

Languages: English

Erratum: Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF (Gene Therapy) (2005) vol. 12 (452-460) 10.1038/sj.gt.3302421)

2/3,K/11 (Item 2 from file: 71) **Links**
Fulltext available through: American Society of Hematology USPTO Full Text Retrieval Options SCIENCEDIRECT
ELSEVIER BIOBASE

02148053 2002228748

Estrogen-dependent E2a/Pbxl myeloid cell lines exhibit conditional differentiation that can be arrested by other leukemic oncoproteins

Sykes D.B.; Kamps M.P.
Address: D.B. Sykes, Department of Molecular Pathology, University of California, San Diego School of Medicine, 9500 Gilman Dr, San Diego, CA 92093-0612 , United States
Email: dsykes@ucsd.edu
Journal : Blood , 98/8 (2308-2318) , 2001 , United States
PUBLICATION DATE: October 15, 2001
CODEN: BLOOA
ISSN: 0006-4971
Document Type: Article
Languages: English **Summary Languages:** English
No. of References: 76

...The cell lines were established by conditional immortalization of primary murine marrow progenitors with an **estrogen** -regulated E2a/Pbxlestrogen **receptor** fusion protein. Clones were identified that proliferated as immortalized blasts in the presence of **estrogen** , and that exhibited granulocytic, monocytic, or bipotential (granulocytic and monocytic) differentiation on **estrogen** withdrawal. Differentiation was normal and terminal as evidenced by morphology, cell surface markers, gene expression... ...differentiation of the cells could be arrested by heterologous oncoproteins including AML1/ETO, PML/RARalpha **PLZF**/RARalpha, Nup98/HoxA9, and other Hox proteins. Furthermore, the study examined the effects of cooperating...

2/3,K/12 (Item 1 from file: 73) **Links**
Fulltext available through: USPTO Full Text Retrieval Options SCIENCEDIRECT
EMBASE

13882758 **EMBASE No:** 2006300624

PLZF regulates Pbx1 transcription and Pbx1-HoxC8 complex leads to androgen-independent prostate cancer proliferation

Kikugawa T.; Kinugasa Y.; Shiraishi K.; Nanba D.; Nakashiro K.-I.; Tanji N. ; Yokoyama M.; Higashiyama S.
Dr. S. Higashiyama, Department of Biochemistry and Molecular Genetics, Ehime University School of Medicine, Shitsukawa, To-on, Ehime 791-0295 Japan
Author Email: shigeki@m.ehime-u.ac.jp
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Number Of References: 38

BACKGROUND. Promyelocytic leukemia zinc finger (**PLZF**) protein, a transcriptional repressor and negative regulator of the cell cycle, has been characterized as a prostatic **androgen**-responsive gene. DU145 cells show **androgen**-independent growth and lack **PLZF** gene expression. METHODS. We analyzed **PLZF**-regulating genes by DNA microarray using DU145 cells infected with LacZ- or **PLZF**-carrying adenoviruses. RESULTS. DNA microarray revealed that Pbx1 is a prominent suppressed gene in **PLZF**-overexpressing DU145 cells. **Androgen receptor** (AR)-expressing DU145 cells recovered **androgen** -dependent **PLZF** expression and subsequent repression of Pbx1 expression. Immunoprecipitation of Pbx1 in DU145 cells revealed a Pbx1-HoxC8 heterocomplex. siRNAs for Pbx1 and HoxC8 knocked downexpression of each, and this suppressed **androgen**-independent cell growth. Double knockdown of both Pbx1 and HoxC8 suppressed cell growth much more significantly. CONCLUSIONS. **Androgen**-independent cell line DU145 cells lack **PLZF** gene expression, resulting in the upregulation of Pbx1 and HoxC8 expression. The Pbx1-HoxC8 heterocomplex may lead to **androgen**-independent growth in prostate cancer. (c) 2006 Wiley-Liss, Inc.

2/3,K/13 (Item 1 from file: 144) **Links**
Pascal

17733282 PASCAL No.: 06-0327020

PLZF regulates pbxl transcription and Pbxl - HoxC8 complex leads to androgen-lndependent prostate cancer proliferation

KIKUGAWA Tadahiko; KINUGASA Yumi; SHIRAISHI Ken; NANBA Daisuke ; NAKASHIRO Koh-Ichi; TANJI Nozomu; YOKOYAMA Masayoshi; HIGASHIYAMA Shigeki

Department of Biochemistryand Molecular Genetics, Ehime University School of Medicine, Shitsukawa, To-on, Ehime, Japan; Department of Urology, Ehime University School of Medicine, Shitsukawa, To-on, Ehime, Japan; Department of Oral and Maxillofacial Surgery, Ehime University School of Medicine, Shitsukawa, To-on, Ehime, Japan; Information and Cell Function, PRESTO, JST , Japan
Journal: The Prostate, 2006 , 66 (10) 1092-1099
Language: English

BACKGROUND. Promyelocytic leukemia zinc finger (**PLZF**) protein, a transcriptional repressor and negative regulator of the cell cycle, has been characterized as a prostatic **androgen**-responsive gene. DU145 cells show **androgen**-independent growth and lack **PLZF** gene expression. METHODS. We analyzed **PLZF** -regulating genes by DNA microarray using DU145 cells infected with LacZ- or **PLZF**-carrying adenoviruses. RESULTS. DNA microarray revealed that Pbxl is a prominent suppressed gene in **PLZF**-overexpressing DU145 cells. **Androgen receptor** (AR)-expressing DU145 cells recovered **androgen** -dependent **PLZF** expression and subsequent repression of Pbxl expression. Immunoprecipitation of Pbxl in DU145 cells revealed a Pbxl-HoxC8 heterocomplex. siRNAs for Pbx1 and HoxC8 knocked downexpression of each, and this suppressed **androgen** -independent cell growth. Double knockdown of both Pbx1 and HoxC8 suppressed cell growth much more significantly. CONCLUSIONS. **Androgen** -independent cell line DU145 cells lack **PLZF** gene expression, resulting in the upregulation of Pbx1 and HoxC8 expression. The Pbxl -HoxC8 heterocomplex may lead to **androgen**-independent growth in prostate cancer.

2/3,K/14 (Item 2 from file: 144) Links
Pascal

16657020 PASCAL No.: 04-0308161

Identification and characterization of PLZF as a prostatic androgen-responsive gene

FENG JIANG; ZHOU WANG
Department of Urology, Northwestern University, Chicago, Illinois, United States; Department of Molecular Pharmacology and Biological Chemistry, and The Robert H. Lurie Comprehensive Cancer Center, Feinberg School of Medicine, Northwestern University, Chicago, Illinois, United States
Journal: The Prostate, 2004
, 59 (4) 426-435
Language: English

BACKGROUND. Promyelocytic leukemia zinc finger protein (**PLZF**) was initially identified by virtue of its fusion with RARa as a result of a...

... 17) chromosomal translocation that occurs in a small subset of acute promyelocytic leukemia (APL) patients. **PLZF** has been reported to have pro-apoptotic and anti-proliferative activity both in vivo and in vitro. METHODS. Using a modified subtractive hybridization, we identified **PLZF** as an **androgen** -responsive gene in the rat ventral prostate. Northern blot and Western blot were used to characterize the regulation of **PLZF** by androgens in LNCaP cells. Stable transfections of **PLZF** in LNCaP cells were performed to assay the effect of **PLZF** overexpression on LNCaP cell proliferation. RESULTS. **PLZF** mRNA was transiently up-regulated by androgens in the regressed ventral prostate of castrated adult rat. **PLZF** was also up-regulated by androgens, at both mRNA and protein levels, in the **androgen** -responsive human prostate cancer cell line LNCaP. **Androgen** induction of **PLZF** mRNA was not inhibited by protein synthesis inhibitor cycloheximide but inhibited by **androgen receptor** antagonist bicalutamide, indicating that **PLZF** is a direct **androgen**-responsive gene. To study the functions of **PLZF** in **androgen** action, LNCaP sublines stably overexpressing **PLZF** were generated. **PLZF** overexpression inhibited LNCaP proliferation either in the presence or absence of **androgen**, which is consistent with the reported anti-proliferative activity of **PLZF**. CONCLUSIONS. The above observations indicate that **PLZF** is an **androgen** -responsive gene with anti-proliferative activity in prostate cancer cells.

2/3,K/15 (Item 1 from file: 266) Links
FEDRIP

00492843
Identifying No.: 137839 ; 0004; 586 **Agency Code:** VA
Dimerization and Dominant Negative Activity of Verb-A

Principal Investigator: Subauste, Jose S., M.D.
Performing Org.: Department of Veterans Affairs, Medical Center Jackson, MS
Sponsoring Org.: Department of Veterans Affairs, Research and Development (15) , 810 Vermont Ave. N.W. , Washington , D.C. 20420 United States of America
Dates: 20001214
Summary: ...blotting, Western blotting, generation and characteriztion of transgenic mice.
CLINICAL RELEVANCE: Mutant forms of nuclear **receptors** have been implicated in a variety of endocrine and neoplastic diseases seen in VA patients, including mutations in TR? and the generalized thyroid hormone resistance syndrome, mutations in the **androgen receptor** and **androgen** resistance syndrome, PML-RAR and **PLZF**-RAR fusion proteins and promyelocytic leukemia, ETO fusion protein and acute myelogenous (M2) leukemia, mutations in the **estrogen receptor** and hormone resistant breast cancers, mutations in the **androgen receptor** and prostate cancer, mutations in thyroid hormone **receptor** and hepatocellular carcinoma. In the majority of these cases, the mutant **receptor** appears to function as a dominant negative form inducing the disease by interfering with the action of the normal **receptor** counterpart. Therefore investigation of v-erbA may lead to important findings that can be applied ...

2/3,K/16 (Item 1 from file: 357) **Links**

Fulltext available through: Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT

Derwent Biotech Res.

0364779 **DBA Accession No.:** 2005-10483

Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF method of mamma carcinoma gene therapy involving gene transfer of an estrogen receptor fusion protein

Author: BULUWELA L; PIKE J; MAZHAR D; KAMALATI T; HART SM; AL-JEHANI R; YAHAYA H; PATEL N; SARWARL N; HEATHCOTE DA; SCHWICKERATH O; PHOENIX F; HILL R; ABOAGYE E; SHOUSHA S; WAXMAN J; LEMOINE NR; ZELENT A; COOMBES RC; ALI S

Corporate Affiliate: Univ London Imperial Coll Sci Technol and Med Univ London Imperial Coll Sci Technol and Med; Univ London; Inst Canc Res

Corporate Source: Buluwela L, Univ London Imperial Coll Sci Technol and Med, Dept Canc Med, Du Cane Rd, London W12 0NN, England

Journal: GENE THERAPY (12, 5, 452-460) 2005

ISSN: 0969-7128

Language: English

Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-alpha and the transcriptional repressor PLZF method of mamma carcinoma gene therapy involving gene transfer of an estrogen receptor fusion protein

Abstract: AUTHOR ABSTRACT - Estrogen receptor alpha (ERalpha) is a ligand-inducible transcription factor that acts to regulate gene expression by binding to palindromic DNA sequence, known as the estrogen response element, in promoters of estrogen-regulated genes. In breast cancer ERalpha plays a central role, where estrogen-regulated gene expression leads to tumor initiation, growth and survival. As an approach to silencing estrogen-regulated genes, we have studied the activities of a fusion protein between ERalpha and the promyelocytic leukemia zinc-finger (PLZF) protein, a transcriptional repressor that acts through chromatin remodeling. To do this, we have developed lines from the estrogen-responsive MCF-7 breast cancer cell line in which the expression of the fusion protein PLZF-ERalpha is conditionally regulated by tetracycline and shows that these feature long-term silencing of the expression of several well-characterized estrogen-regulated genes, namely pS2, cathepsin-D and the progesterone receptor. However, the estrogen-regulated growth of these cells is not inhibited unless PLZF-ERalpha expression is induced, an observation that we have confirmed both in vitro and in vivo. Taken together, these results show that PLZF-ERalpha is a potent repressor of estrogen-regulated gene expression and could be useful in distinguishing estrogen-regulated genes required for the growth of breast cancer cells. (9 pages)

2/3,K/17 (Item 2 from file: 357) **Links**

Fulltext available through: Ebsco Host EJS (Electronic Journals Service) Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT

Derwent Biotech Res.

0357546 **DBA Accession No.:** 2005-03250

Silencing of androgen-regulated genes using a fusion of AR with the PLZF transcriptional repressor androgen-regulated protein gene silencing using transcription repressor for gene therapy

Author: PIKE J; HOLMES D; KAMALATI T; DAVIES D; TOLHURST R; MAZHAR D; FISHPOOL S; AL-JEHANI R; WAXMAN J; ZELENT A; LEMOINE NR; ALI S; BULUWELA L

Corporate Affiliate: Univ London Imperial Coll Sci Technol and Med Canc Res UK Inst Canc Res

Corporate Source: Ali S, Univ London Imperial Coll Sci Technol and Med, Dept Canc Med, Du Cane Rd, London W12 0NN, England

Journal: ONCOGENE (23, 45, 7561-7570) 2004

ISSN: 0950-9232

Language: English

Abstract: AUTHOR ABSTRACT - The **androgen receptor** (AR) is a member of the nuclear **receptor** superfamily of ligand-activated transcription factors and plays a key role in the development and... ...regulated genes, based on the properties of the transcriptional repressor promyelocytic leukamia zinc-finger protein (**PLZF**). In order to do this, we have made a fusion protein between **PLZF** and AR, named **PLZF**-AR, and show that **PLZF**-AR is able to bring about silencing of genomically encoded AR-regulated genes and inhibit the **androgen**-regulated growth of LNCaP prostate cancer cells. Together, our results show that this strategy is...

2/3,K/18 (Item 3 from file: 357) Links
Derwent Biotech Res.

0309775 **DBA Accession No.:** 2003-11560 **PATENT**

Suppressing the expression of a selected endogenous gene in a eukaryotic cell, useful in medicine, comprises introducing into the cell a molecule comprising a nucleic acid binding portion or a polynucleotide encoding the molecule vector-mediated histone deacetylation complex or promyelocytic leukemia zinc finger N-CoR- or SMRT-binding domain gene transfer and expression in host cell for gene therapy

Author: BULUWELA L; HOLMES D; KAMALATI T; WAXMAN J; ALI S
Patent Assignee: GENE EXPRESSION TECHNOLOGIES LTD 2003
Patent Number: WO 2003010308 **Patent Date:** 20030206 **WPI Accession No.:** 2003-248079 (200324)
Priority Application Number: GB 200117964 **Application Date:** 20010724
National Application Number: WO 2002GB3336 **Application Date:** 20020719
Language: English
Abstract: ...b) all or a N-CoR- or SMRT-binding part of promyelocytic leukemia zinc finger (**PLZF**); or (c) an enzymatically active part of a HDAC. The component of the HDAC complex... ...that binds to or facilitates the recruitment of a HDAC complex is any one of **PLZF**, N-CoR, SMRT, Sin3, SAP18, SAP30, or HDAC. The nucleic acid binding portion is a... ...plant or animal genome; (d) all or a DNA binding part of a steroid hormone **receptor** protein or other nuclear **receptor** DNA binding protein; or (e) all or a DNA-binding portion of **estrogen receptor** (ER) or all or a DNA-binding portion of **androgen receptor** (AR). The method further comprises exposing the cell to the ligand. The binding of the... ...and ligand binding portion are derivable from different polypeptides, e.g. from different steroid hormone **receptors**. Suppressing the expression of a selected gene in a eukaryotic cell comprises introducing into the... ...plant cell. ACTIVITY - Cytostatic; Virucide; Anti-HIV. A retroviral vector was produced which encodes a **PLZF**-ER or **PLZF**-AR-ER fusion protein. Following packaging, the recombinant retrovirus was transduced into breast cancer cells in situ and **estrogen receptor**-mediated transcription was suppressed selectively in breast cells. The retroviral vector was administered into the site of breast tumor. Retroviral RNA was taken up by the breast cancer cells and **estrogen receptor**-mediated transcription was suppressed selectively in breast cells. MECHANISM OF ACTION - ...genes such as HIV. EXAMPLE - MCF7-Tet Off line JP23 cells were seeded in an **estrogen** free-media containing doxytetracycline (TET, 1 micro-g/ml) and maintained for 2 days. Cell culture media were then altered so that **estrogen** -regulated growth could be assessed in the presence (no TET) and absence (with TET) of promyelocytic leukemia zinc finger (**PLZF**)- **estrogen receptor** (ER). **Estrogen** regulated growth was inhibited in JP23 cells in the presence of **PLZF**-ER expression. Cells expressing **PLZF**-ER for 4 days were re-treated with TET and under these conditions **PLZF**-ER expression was lost within 24 hours. Over the remaining 5 days of the assay, the cells showed little growth indicating a greatly reduced growth response to **estrogen**. (89 pages)

2/3,K/19 (Item 1 from file: 399) **Links**

Fulltext available through: Nature American, Inc. (Publisher Group) USPTO Full Text Retrieval Options SCIENCEDIRECT

CA SEARCH(R)

143400696 **CA:** 143(22)400696b JOURNAL

Inhibiting estrogen responses in breast cancer cells using a fusion protein encoding estrogen receptor-.alpha. and the transcriptional repressor PLZF. (Erratum to document cited in CA142:387094)

Author: Buluwela, L.; Pike, J.; Mazhar, D.; Kamalati, T.; Hart, S. M.; Al-Jehani, R.; Yahaya, H.; Patel, N.; Sarwarl, N.; Heathcote, D. A.; Schwickerath, O.; Phoenix, F.; Hill, R.; Aboagye, E.; Shousha, S.; Waxman, J.; Lemoine, N. R. ; Zelent, A.; Coombes, R. C.; Ali, S.

Location: Department of Cancer Medicine, Imperial College London, London, UK,

Journal: Gene Ther.

Date: 2005

Volume: 12 **Number:** 10 **Pages:** 862

CODEN: GETHEC

ISSN: 0969-7128

Language: English

Publisher: Nature Publishing Group

2/3,K/20 (Item 2 from file: 399) **Links**
Fulltext available through: USPTO Full Text Retrieval Options SCIENCEDIRECT
CA SEARCH(R)

140000921 **CA:** 140(1)921k JOURNAL
Specific regulation of lipocalin-type prostaglandin D synthase in mouse heart by estrogen receptor .beta.
Author: Otsuki, Michio; Gao, Hui; Dahlman-Wright, Karin; Ohlsson, Claes; Eguchi, Naomi; Urade, Yoshihiro; Gustafsson, Jan-Ake
Location: Department of Biosciences at Novum, Karolinska Institutet, SE-14157, Huddinge, Swed.
Journal: Mol. Endocrinol.
Date: 2003
Volume: 17 **Number:** 9 **Pages:** 1844-1855
CODEN: MOENEN
ISSN: 0888-8809
Language: English
Publisher: Endocrine Society

2/3,K/21 (Item 3 from file: 399) **Links**
CA SEARCH(R)

134096209 **CA:** 134(8)96209j PATENT
Suppression of gene expression using fusion proteins of DNA-binding and chromatin inactivation domains
Inventor (Author): Buluwela, Lakjaya; Ali, Simak
Location: UK,
Assignee: Imperial College Innovations Limited
Patent: PCT International ; WO 200102019 A2 **Date:** 20010111
Application: WO 2000GB2497 (20000628) *GB 9915126 (19990630)
Pages: 65 pp.
CODEN: PIXXD2
Language: English
Patent Classifications:
Class: A61K-048/00A
Designated Countries: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
Designated Regional: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3,K/22 (Item 1 from file: 35) **Links**
Dissertation Abs Online

02020026 ORDER NO: AADAA-I3132545
Mechanism of androgen action in the prostate: Identification and analysis of androgen-responsive genes

Author: Jiang, Feng
Degree: Ph.D.
Year: 2004
Corporate Source/Institution: Northwestern University (0163)
Source: Volume 6505B of Dissertations Abstracts International.
PAGE 2244 . 159 PAGES

Androgens are required for the structural and functional integrity of the prostate. **Androgen** action is intimately involved in the pathogenesis of two major prostate diseases, benign prostatic hyperplasia (BPH) and prostate carcinoma (CaP). Androgens regulate gene expression in the prostate through the **androgen receptor** (AR), a ligand-dependent transcription factor. To understand the molecular and cellular mechanisms of **androgen** action in the prostate, I used both cDNA subtractive hybridizations and microarray to comprehensively identify ... ...trafficking, secretions, cell cycle and apoptosis, and structural and extracellular proteins. I have characterized four **androgen** -responsive genes: FPPS, **PLZF**, GADD45γ, and U19. FPPS is abundantly expressed and regulated by androgens in the rat prostatic epithelial cells. Both **PLZF** and GADD45γ are found to be growth-suppressive to prostate cancer cells, suggesting that androgens might activate a signaling pathway to counteract the **androgen** -induced cell proliferation pathway. U19, a novel **androgen** -responsive apoptosis inducer, is also growth-suppressive to prostate cancer. Therefore, U19 was chosen for... ...activity of U19. In conclusion, my thesis established a foundation for future mechanistic study of **androgen** action and provided new insights into the roles played by androgens in the prostate.